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1	2313	(435/7.9,7.93).CCLS.	USPAT; EPO	2004/10/21 07:50
2	3545	(436/514,518).CCLS.	USPAT; EPO	2004/10/21 07:50
3	112	steinbeck.in.	USPAT; EPO	2004/10/21 07:51
4	2	chlorinat\$4 same arthritis	USPAT; EPO	2004/10/21 07:52
5	6	chlorinat\$4 same arthritis	USPAT; US-PGPUB; EPO; DERWENT	2004/10/21 07:53
6	6	cartilage same chlorinat\$4	USPAT; US-PGPUB; EPO; DERWENT	2004/10/21 07:55
7	1	cartilage same chlorinat\$4 same antibody	USPAT; US-PGPUB; EPO; DERWENT	2004/10/21 07:55
8	6	arthritis same chlorinat\$4	USPAT; US-PGPUB; EPO; DERWENT	2004/10/21 07:55

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=> (chlorinated peptide) and osteoarthritis

L1	0	FILE AGRICOLA
L2	0	FILE BIOTECHNO
L3	0	FILE CONFSCI
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L5	0	FILE IMSDRUGCONF
L6	0	FILE LIFESCI
L7	0	FILE MEDICONF
L8	0	FILE PASCAL

TOTAL FOR ALL FILES

L9	0	(CHLORINATED PEPTIDE) AND OSTEOARTHRITIS
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=> osteoarthritis

L10	313	FILE AGRICOLA
L11	1713	FILE BIOTECHNO
L12	458	FILE CONFSCI
L13	60	FILE HEALSAFE
L14	10	FILE IMSDRUGCONF
L15	1361	FILE LIFESCI
L16	141	FILE MEDICONF
L17	9984	FILE PASCAL

TOTAL FOR ALL FILES

L18	14040	OSTEOARTHRITIS
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=> (chlorinated or chlorination) and arthritis

L19	0	FILE AGRICOLA
L20	6	FILE BIOTECHNO
L21	0	FILE CONFSCI
L22	0	FILE HEALSAFE

L23 0 FILE IMSDRUGCONF
L24 5 FILE LIFESCI
L25 0 FILE MEDICONF
L26 4 FILE PASCAL

TOTAL FOR ALL FILES

L27 15 (CHLORINATED OR CHLORINATION) AND ARTHRITIS

=> dup rem

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PROCESSING COMPLETED FOR L27

L28 10 DUP REM L27 (5 DUPLICATES REMOVED)

=> d l28 ibib abs

L28 ANSWER 1 OF 10 LIFESCI COPYRIGHT 2004 CSA on STN

ACCESSION NUMBER: 2004:64518 LIFESCI

TITLE: Risk analysis of drinking water microbial contamination
versus disinfection by-products (DBPs)

AUTHOR: Ashbolt, N.J.

CORPORATE SOURCE: CRC for Water Quality and Treatment, School of Civil and
Environmental Engineering, UNSW, Sydney, NSW 2052,
Australia; E-mail: n.ashbolt@unsw.edu.au

SOURCE: Toxicology, (20040500) vol. 198, no. 1-3, pp. 255-262.
ISSN: 0300-483X.

DOCUMENT TYPE: Journal

FILE SEGMENT: X

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Managing the provision of safe drinking water has a renewed focus in light of the new World Health Organization (WHO) water safety plans. Risk analysis is a necessary component to assist in selecting priority hazards and identifying hazardous scenarios, be they qualitative to quantitative assessments. For any approach, acute diarrhoeal pathogens are often the higher risk issue for municipal water supplies, no matter how health burden is assessed. Furthermore, potential sequelae (myocarditis, diabetes, reactive arthritis and cancers) only further increase the potential health burden of pathogens; despite the enormous uncertainties in determining pathogen exposures and chemical dose-responses within respective microbial and chemical analyses. These interpretations are currently being improved by Bayesian and bootstrapping approaches to estimate parameters for stochastic assessments. A case example, covering the health benefits of ozonation for *Cryptosporidium* inactivation versus potential cancers from bromate exposures, illustrated the higher risks from a pathogen than one of the most likely disinfection by-products (DBPs). Such analyses help justify the industries long-held view of the benefits of multiple barriers to hazards and that microbial contamination of water supplies pose a clear public health risk when treatment is inadequate. Therefore, efforts to reduce potential health risks from DBP must not compromise pathogen control, despite socio-political issues.

=> d l28 ibib abs total

L28 ANSWER 1 OF 10 LIFESCI COPYRIGHT 2004 CSA on STN

ACCESSION NUMBER: 2004:64518 LIFESCI

TITLE: Risk analysis of drinking water microbial contamination
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AUTHOR: Ashbolt, N.J.

CORPORATE SOURCE: CRC for Water Quality and Treatment, School of Civil and
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Australia; E-mail: n.ashbolt@unsw.edu.au
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ISSN: 0300-483X.

DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English

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L28 ANSWER 2 OF 10 LIFESCI COPYRIGHT 2004 CSA on STN DUPLICATE 1

ACCESSION NUMBER: 2004:64507 LIFESCI
TITLE: Microbial contamination of drinking water and disease outcomes in developing regions
AUTHOR: Ashbolt, N.J.
CORPORATE SOURCE: School of Civil and Environmental Engineering, University of New South Wales, Sydney, NSW 2052, Australia; E-mail: N.Ashbolt@unsw.edu.au
SOURCE: Toxicology, (20040500) vol. 198, no. 1-3, pp. 229-238.
ISSN: 0300-483X.

DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Drinking water is a major source of microbial pathogens in developing regions, although poor sanitation and food sources are integral to enteric pathogen exposure. Gastrointestinal disease outcomes are also more severe, due to under-nutrition and lack of intervention strategies in these regions. Poor water quality, sanitation and hygiene account for some 1.7 million deaths a year world-wide (3.1% of all deaths and 3.7% of all DALY's), mainly through infectious diarrhoea. Nine out of 10 such deaths are in children and virtually all of the deaths are in developing countries. Major enteric pathogens in these children include: rotavirus, *Campylobacter jejuni*, enterotoxigenic *Escherichia coli*, *Shigella* spp. and *Vibrio cholerae* O1, and possibly enteropathogenic *E. coli*, *Aeromonas* spp. *V. cholerae* O139, enterotoxigenic *Bacteroides fragilis*, *Clostridium difficile* and *Cryptosporidium parvum*. All except the latter are easily control by **chlorination** of water, but recontamination of treated water is a huge problem. Emerging environmental pathogens, such as *Helicobacter pylori* and *Burkholderia pseudomallei*, may well be of significance in some regions. In adults, much less is understood of various sequelae such as myocarditis, diabetes, reactive **arthritis** and cancers some months-years after initial infections. So in addition to the traditional pathogens (helminths, *Entamoeba*

histolytica, Giardia lamblia hepatitis A and E) various enteroviruses, C. jejuni and H. pylori are emerging issues in adults.

L28 ANSWER 3 OF 10 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2003:36682362 BIOTECHNO
TITLE: Selective inhibition of cyclooxygenase 2-generated
prostaglandin E.sub.2 synthesis in rheumatoid
arthritis synoviocytes by taurine chloramine
AUTHOR: Kontny E.; Rudnicka W.; Kowalczewski J.; Marcinkiewicz
J.; Maslinski W.
CORPORATE SOURCE: Dr. E. Kontny, Department of Pathophysiology,
Institute of Rheumatology, Spartanska 1, 02-637
Warsaw, Poland.
E-mail: zpatiir@warman.com.pl
SOURCE: Arthritis and Rheumatism, (01 JUN 2003), 48/6
(1551-1555), 17 reference(s)
CODEN: ARHEAW ISSN: 0004-3591
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AN 2003:36682362 BIOTECHNO

AB Objective. To investigate the effects of taurine chloramine (Tau-Cl), a
chlorinated derivative of the amino acid taurine, on the
expression of cyclooxygenase (COX) isoenzymes and prostaglandin E.sub.2
(PGE.sub.2) synthesis in rheumatoid **arthritis** (RA)
fibroblast-like synoviocytes (FLS). Methods. FLS, isolated from the
synovial tissue of RA patients, were treated in vitro with either
interleukin-1 β (IL-1 β ; 1 ng/ml) alone or together with 200-500
 μ M Tau-Cl. The expression of COX isoenzymes was evaluated at both the
protein (Western blotting) and the messenger RNA (mRNA) (reverse
transcriptase-polymerase chain reaction) levels. The concentration of
PGE.sub.2 was measured by competitive acetylcholinesterase enzyme
immunoassay. Results. Resting FLS expressed mRNA encoding both COX-1 and
COX-2, but only COX-1 was present at the protein level. These cells
produced negligible amounts of PGE.sub.2. Upon stimulation with
IL-1 β , elevation of COX-2, but not COX-1, mRNA and protein preceded
the enhancement of PGE.sub.2 synthesis. In the presence of 300-400 μ M
Tau-Cl, significant inhibition of IL-1 β -triggered COX-2 mRNA and
protein, and a related decrease in PGE.sub.2 production, was observed. In
contrast, no significant changes in COX-1 mRNA and protein levels were
noted. Conclusion. Tau-Cl inhibits IL-1 β -triggered elevation of
COX-2 and generation of PGE.sub.2 by RA FLS. These results expand the
spectrum of known antiinflammatory activities of this compound.

L28 ANSWER 4 OF 10 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2000:30819247 BIOTECHNO
TITLE: **Chlorination** of pyridinium compounds.
Possible role of hypochlorite, N-chloramines, and
chlorine in the oxidation of pyridinoline cross-links
of articular cartilage collagen type II during acute
inflammation
AUTHOR: Daumer K.M.; Khan A.U.; Steinbeck M.J.
CORPORATE SOURCE: M.J. Steinbeck, Dept. of Orthopaedic Surgery, Thomas
Jefferson University, Curtis Bldg., 1015 Walnut St.,
Philadelphia, PA 19107, United States.
E-mail: marla.steinbeck@mail.tju.edu
SOURCE: Journal of Biological Chemistry, (03 NOV 2000), 275/44
(34681-34692), 72 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English

SUMMARY LANGUAGE: English

AN 2000:30819247 BIOTECHNO

AB Reactive oxygen species produced by activated neutrophils and monocytes are thought to be involved in mediating the loss of collagen and other matrix proteins at sites of inflammation. To evaluate their potential to oxidize the pyridinoline (Pyd) cross-links found in collagen types I and II, we reacted hydrogen peroxide (H.sub.2O.sub.2), hypochlorous acid/hypochlorite (HOCl/OCl.sup.-), and singlet oxygen (O.sub.2(.sup.1Ag)) with the Pyd substitutes, pyridoxamine dihydrochloride and vitamin B.sub.6, which share the same chemical structure and spectral properties of Pyd cross-links. Neither H.sub.2O.sub.2 (125-500 µM) nor O.sub.2(.sup.1Ag) (10-25 µM) significantly changed the spectral properties of pyridoxamine or vitamin B.sub.6. Reaction of HOCl/OCl.sup.- (12.5-50 µM) with pyridoxamine at pH 7.2 resulted in a concentration-dependent appearance of two new absorbance peaks and a decrease in fluorescence at 400 nm (excitation 325 nm). The new absorbance peaks correlated with the formation of an N-chloramine and the product of its subsequent reaction with pyridoxamine. In contrast, the extent to which HOCl reacted with vitamin B.sub.6, which lacks a primary amine group, was variable at this pH. At lysosomal pH 5.5, Cl.sub.2/HOCl/OCl.sup.- reacted with both pyridoxamine and vitamin B.sub.6. Four of the **chlorinated** products of this reaction were identified by gas chromatography-mass spectrometry and included 3-chloropyridinium, an aldehyde, and several **chlorinated** products with disrupted rings. To evaluate the effects of Cl.sub.2/HOCl/OCl.sup.- on Pyd cross-links in collagen, we exposed bone collagen type I and articular cartilage type II to HOCl. Treatment of either collagen type with HOCl at pH 5.0 or 7.2 resulted in the oxidation of amine groups and, for collagen type II, the specific decrease in Pyd cross-link fluorescence, suggesting that during inflammation both oxidations may be used by neutrophils and monocytes to promote the loss of matrix integrity.

L28 ANSWER 5 OF 10 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN DUPLICATE

ACCESSION NUMBER: 2000:30978768 BIOTECHNO

TITLE: Inflammatory properties of IgG modified by oxygen radicals and peroxynitrite

AUTHOR: Uesugi M.; Yoshida K.; Jasin H.E.

CORPORATE SOURCE: Dr. H.E. Jasin, Univ. of Arkansas for Med. Sciences, Mail Slot 509, 4301 West Markham, Little Rock, AR 72205, United States.

E-mail: jasin@exchange.uams.edu

SOURCE: Journal of Immunology, (01 DEC 2000), 165/11 (6532-6537), 28 reference(s)

CODEN: JOIMA3 ISSN: 0022-1767

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 2000:30978768 BIOTECHNO

AB In inflammatory **arthritis**, there is evidence indicating that the affected tissues produce large amounts of oxygen-free radicals and NO. Herein, we examine the biologic effects of exposure of IgG to hypochlorous acid (HOCl) and peroxynitrite (ONOO). The concentrations of IgG modified by **chlorination** and nitrosation were measured in synovial fluids from inflammatory and noninflammatory **arthritis**. Human IgG was exposed to increasing concentrations of HOCl and ONOO, and the resulting products were tested for complement component binding; binding to FcγRI; activation of polymorphonuclear neutrophils; effect on the Ab-combining site of Abs; and in vivo inflammatory activity in a rabbit model of acute **arthritis**. Rheumatoid synovial fluids contained significantly greater concentrations of nitrosated and **chlorinated** IgG compared with osteoarthritic specimens. In vitro

exposure of human IgG to HOCl and ONOO resulted in a concentration-dependent decrease in C3 and Clq fixation. The decrease in Fc domain-dependent biologic functions was confirmed by competitive binding studies to the FcγRI of U937 cells. HOCl-treated IgG monomer was 10 times less effective in competing for binding compared with native IgG, and ONOO-treated IgG was 2.5 times less effective. The modified IgGs were also ineffective in inducing synthesis of H.sub.2O.sub.2 by human PMN. The Ag-binding domains of IgG also showed a concentration-dependent decrease in binding to Ag. The ability of the modified IgGs to induce acute inflammation in rabbit knees decreased 20-fold as gauged by the intensity of the inflammatory cell exudates. These studies clarify the modulating role of biological oxidants in inflammatory processes in which Ag-autoantibody reactions and immune complex pathogenesis may play an important role.

ANSWER 6 OF 10 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE

SESSION NUMBER: 1999:30327171 BIOTECHNO
 TITLE: Taurine chloramine inhibition of cell proliferation and cytokine production by rheumatoid arthritis fibroblast-like synoviocytes
 AUTHOR: Kontny E.; Grabowska A.; Kowalczewski J.; Kurowska M.; Janicka I.; Marcinkiewicz J.; Maslinski W.
 ORIGINATE SOURCE: Dr. E. Kontny, Dept. of Pathophysiology/Immunology, Institute of Rheumatology, Spartanska 1, 02-637 Warsaw, Poland.
 SOURCE: Arthritis and Rheumatism, (1999), 42/12 (2552-2560), 40 reference(s)
 CODEN: ARHEAW ISSN: 0004-3591
 DOCUMENT TYPE: Journal; Article
 COUNTRY: United States
 LANGUAGE: English
 PRIMARY LANGUAGE: English

1999:30327171 BIOTECHNO

Objective. To examine whether taurine (Tau) or its physiologic chlorinated derivative, taurine chloramine (Tau-Cl), affects proliferation of, and proinflammatory cytokine (interleukin-6 [IL-6] and IL-8) production by, fibroblast-like synoviocytes (FLS) isolated from rheumatoid arthritis (RA) patients. Methods. FLS, isolated from the synovial tissue of 19 RA patients and cultured in vitro for 3-6 passages, were stimulated with the recombinant human cytokines IL-1β (1 ng/ml), tumor necrosis factor α (TNFα; 10 ng/ml), or IL-17 (10 ng/ml) in the presence of either Tau or Tau-Cl, which were added at concentrations of 50-500 μM. Tau and Tau-Cl were added simultaneously with, 2 hours before, or 24 hours after the stimuli. The concentrations of IL-6 and IL-8 were determined in culture supernatants using specific enzyme-linked immunosorbent assays. Proliferation of FLS was estimated on the basis of 3H-thymidine incorporation into the cells, which were cultured for 72 hours in the presence of recombinant human basic fibroblast growth factor (bFGF) (1 ng/ml) and Tau or Tau-Cl, which were added simultaneously at the beginning of the culture. Results. Cultured in vitro, RA FLS spontaneously secreted low levels of IL-6 and IL-8, but when RA FLS were stimulated with IL-1β, TNFα, or IL-17, significantly higher amounts of IL-6 and IL-8 were produced. Tau-Cl, but not Tau, inhibited cytokine-triggered synthesis of IL-6 (50% inhibitory concentration [IC.sub.50] .sim.225 μM) and IL-8 (IC.sub.50 .sim.450 μM) when added simultaneously with the stimuli. However, IL-17 induced production of IL-8 was not affected by Tau-Cl. In the cells prestimulated with IL-1β for 24 hours, Tau-Cl still inhibited synthesis of IL-6, but did not affect IL-8 production. Moreover, Tau-Cl inhibited spontaneous and bFGF-triggered proliferation of FLS in a dose-dependent manner. Neither Tau nor Tau-Cl affected cell viability. Conclusion. The results of these studies demonstrate that Tau-Cl inhibits production of proinflammatory cytokines by RA FLS, as well as

proliferation of these cells. Thus, Tau-Cl may act as a physiologic modulator of FLS functions related to their pathogenic role in RA.

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ACCESSION NUMBER: 1996-0308374 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): NMR studies on human, pathologically changed synovial fluids : Role of hypochlorous acid
AUTHOR: SCHILLER J.; ARNHOLD J.; SONNTAG K.; ARNOLD K.
CORPORATE SOURCE: Institute of Medical Physics and Biophysics, Medical Department, University of Leipzig, Leipzig, Germany, Federal Republic of
SOURCE: Magnetic resonance in medicine, (1996), 35(6), 848-853, 30 refs.
ISSN: 0740-3194 CODEN: MRMEEN
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-20644, 354000043754580090

AN 1996-0308374 PASCAL

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AB Recently, it has been reported that hypochlorous acid (HOCl), a special product of neutrophil myeloperoxidase, degrades N-acetyl groups of N-acetylglucosamine, chondroitin sulfate, hyaluronic acid, and minced articular cartilage via a transient product to acetate. This work concerns ¹H NMR investigations of synovial fluids of patients with rheumatoid arthritis (RA). Synovial fluids of patients with severe forms of this disease are characterized by enhanced ¹H NMR signals for N-acetyl groups (eqvsim.2.0 ppm) and acetate (1.90 ppm) and the appearance of a broad but less intense signal at 2.35 ppm. It is likely that this signal corresponds to the transient, chlorinated product of degradation of N-acetyl groups by hypochlorous acid. Moreover, ¹H NMR signal intensities of N-acetyl groups and acetate strongly correlate with the myeloperoxidase activities in synovial fluids from patients with rheumatoid arthritis. These results have been confirmed by treatment of native sheep synovial fluid with sodium hypochlorite, resulting in the formation of the same resonances as observed in pathologically changed synovial fluids from humans. Thus, it is concluded that HOCl plays an important role for the cartilage degradation during rheumatoid arthritis.

L28 ANSWER 8 OF 10 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 1995:25328885 BIOTECHNO
TITLE: NMR studies of the action of hypochlorous acid on native pig articular cartilage
AUTHOR: Schiller J.; Arnhold J.; Arnold K.
CORPORATE SOURCE: Inst. Medizinische Physik/Biophysik, Universitätsbereich Medizin, Universität Leipzig, Liebigstrasse 27, D-04103 Leipzig, Germany.
SOURCE: European Journal of Biochemistry, (1995), 233/2 (672-676)
CODEN: EJBCAI ISSN: 0014-2956
DOCUMENT TYPE: Journal; Article
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: English

AN 1995:25328885 BIOTECHNO

AB The action of sodium hypochlorite on pig articular cartilage was studied by ¹H-NMR spectroscopy to model some aspects of degradation processes of cartilage during rheumatoid arthritis. Two effects

of NaOCl on cartilage polysaccharides have been observed. Hypochlorous acid causes an enhanced release of oligomeric polysaccharides from cartilage. The second effect concerns the degradation of N-acetyl side chains of carbohydrates to acetate via a **chlorinated** transient product. Signal intensities for N-acetyl groups (.sim. 2.0 ppm) increase-during the first 2 h of incubation of cartilage with NaOCl. Then they decrease again. However, acetate (1.90 ppm) as the final product of degradation of N-acetyl side chains increases continuously over the period of incubation with NaOCl. In addition to polysaccharides, effects of NaOCl were only observed in cartilage samples on amino acids like alanine. The alanine resonance disappeared already at NaOCl concentrations where only small effects on cartilage polysaccharides have been observed.

L28 ANSWER 9 OF 10 LIFESCI COPYRIGHT 2004 CSA on STN

ACCESSION NUMBER: 96:69837 LIFESCI

TITLE: The action of hypochlorous acid on polymeric components of cartilage

AUTHOR: Schiller, J.; Arnhold, J.*; Gruender, W.; Arnold, K.

CORPORATE SOURCE: Institut Medizinische Physik und Biophysik, Universitaet Leipzig, Liebigstr. 27, 04103 Leipzig, FRG

SOURCE: BIOL. CHEM. HOPPE-SEYLER, (1994) vol. 375, no. 3, pp. 167-172.
ISSN: 0177-3593.

DOCUMENT TYPE: Journal

FILE SEGMENT: T; F

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The action of sodium hypochlorite on N-acetylglucosamine, N-acetylgalactosamine, chondroitinsulfate and hyaluronic acid was studied by super(1)H-nuclear magnetic resonance (super(1)H-NMR) in order to model some aspects of degradation processes caused by neutrophils on carbohydrate polymers of cartilage in rheumatoid **arthritis**. N-Acetyl side groups of carbohydrate monomers and chondroitinsulfate yield a resonance at 2.01-2.04 ppm in proton NMR-spectra. This resonance is observed in hyaluronic acid solutions only after a prolonged incubation to yield shorter polymeric chains. Sodium hypochlorite causes a continuous decrease of the line for N-acetyl groups. Two new resonances appear in the super(1)H-NMR spectra. An intermediate product, assumed as a **chlorinated** product of N-acetyl side chains, shows a chemical shift of about 2.35 ppm. This intermediate is hydrolyzed to a carbohydrate ring and acetate (1.90 ppm). Sodium hypochlorite acts in all systems investigated mainly on N-acetyl groups. Only small effects on the carbohydrate ring were found under our experimental conditions.

L28 ANSWER 10 OF 10 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1987:17039034 BIOTECHNO

TITLE: The effect of D-penicillamine on myeloperoxidase: Formation of Compound III and inhibition of the chlorinating activity

AUTHOR: Cuperus R.A.; Hoogland H.; Wever R.; Muijsers A.O.

CORPORATE SOURCE: Laboratory of Biochemistry, University of Amsterdam, 1000 HD Amsterdam, Netherlands.

SOURCE: Biochimica et Biophysica Acta - Protein Structure and Molecular Enzymology, (1987), 912/1 (124-131)
CODEN: BBAEDZ

DOCUMENT TYPE: Journal; Article

COUNTRY: Netherlands

LANGUAGE: English

AN 1987:17039034 BIOTECHNO

AB The inhibitory effect of the anti-arthritic drug D-penicillamine on the formation of hypochlorite (HOCl) by myeloperoxidase from H.sub.20.sub.2 and Cl.sup.- was investigated. When D-penicillamine was added to myeloperoxidase under turnover conditions, Compound III was formed, the

superoxide derivative of the enzyme. Compound III was not formed when D-penicillamine was added in the presence of EDTA or in the absence of oxygen. However, when H.sub.2O.sub.2 was added to myeloperoxidase, D-penicillamine and EDTA, Compound III was formed. Therefore it is concluded that formation of Compound III is initiated by metal-catalysed oxidation of the thiol group of this anti-arthritic drug, resulting in formation of superoxide anions. Once Compound III is formed, a chain reaction is started via which the thiol groups of other D-penicillamine molecules are oxidized to disulphides. Concomitantly, Compound I of myeloperoxidase would be reduced to Compound II and superoxide anions would be generated from oxygen. This conclusion is supported by experiments which showed that formation of Compound III of myeloperoxidase by D-penicillamine depended on the chloride concentration. Thus, an enzyme intermediate which is active in **chlorination** (i.e. Compound I) participated in the generation of superoxide anions from the anti-arthritic drug. From the results described in this paper it is proposed that D-penicillamine may exert its therapeutic effect in the treatment of rheumatoid **arthritis** by scavenging HOCl and by converting myeloperoxidase to Compound III, which is inactive in the formation of HOCl.